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EXAMINER	
PRIEBE, SCOTT DAVID	
ART UNIT	**PAPER NUMBER**
1632	

DATE MAILED: 04/13/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/700,843	LUKACSOVICH ET AL.	
	Examiner	Art Unit	
	Scott D. Priebe, Ph.D.	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *13 January 2005*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-15* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☒ Claim(s) *4* is/are allowed.

6)☒ Claim(s) *1-3,5-8 and 10-15* is/are rejected.

7)☒ Claim(s) *9* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

A request for continued examination under 37 CFR 1.114 was filed in this application after appeal to the Board of Patent Appeals and Interferences, but prior to a decision on the appeal. Since this application is eligible for continued examination under 37 CFR 1.114 and the fee set forth in 37 CFR 1.17(e) has been timely paid, the appeal has been withdrawn pursuant to 37 CFR 1.114 and prosecution in this application has been reopened pursuant to 37 CFR 1.114. Applicant's submission filed on 1/13/05 has been entered.

The examiner of your application in the PTO has changed. To aid in correlating any papers for this application, all further correspondence regarding this application should be directed to Primary Examiner Scott D. Priebe, Ph.D., Group Art Unit 1632.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Claim 9 was indicated as being rejected on the PTO-326 in the Office action of 12/16/03 and allowed in the Advisory action of 7/9/04. Claim 4 was indicated as being allowed in the in the Office action of 12/16/03 and objected to in the Advisory action. The Office action of 12/16/03 did not contain a rejection of claim 9 nor an objection of claim 4, nor did the Advisory action contain an objection to claim 4. As indicated in this Office action, claim 4 is allowed, and claim 9 is newly rejected.

The rejection under 35 USC 103 is withdrawn in part because of Applicant's arguments in the replies of 9/29/03 and 6/16/04, and also because the combination of prior art references

cited in the rejection do not include or suggest all the limitations present in the independent claims. The claims require a dual 5'/3' gene trap cassette that includes a 5' gene trap construct, a drug resistance gene (which is for selection of transformants, and not for gene trapping) and a 3' gene trap construct. Sands does not describe dual gene trap cassettes, and the reporter gene for the gene trap is also the selection marker for identifying transformants, i.e. there is no need for a second selection marker. The prior art closest to the claimed vector is represented by pGawB described in Brand et al. (Development 118: 410-415, 1993) in Fig. 2, which was used by Gustafson et al. pGawB comprises recombinant P-element with a Gal4 reporter gene under control of a weak promoter and a mini-white gene that includes the white promoter and transcription termination sequences. In this construct, the Gal4 reporter is used to trap enhancer elements, not promoters. Insertion of the P-element into the 5' UTR of an endogenous gene provides an enhancer(s) that increases expression of Gal4 from the weak promoter. The white gene in pGawB is analogous to the drug resistance gene of the claimed vector, i.e. it is used to select transformants having pGawB. In order to arrive at the claimed vector, one would have to make three separate modifications to pGawB. First, the weak promoter would be replaced with a splice acceptor and stop/start sequence for trapping promoters and not just enhancers. Second, a drug resistance gene would have to be inserted into the P-element. Third, the white gene transcription termination sequences would have to be replaced with a splice donor sequence. While the first modification might be obvious over Sands in order to convert pGawB into a 5' gene (or promoter) trap vector for *Drosophila*, analogous to VICTR1 of Sands, there would be no motivation to make both the second and third modifications as well. The closest Sands comes to a dual gene trap vector is illustrated by VICTR2, which is simply an artificial exon. Insertion

of the vector into an intron of a gene cause insertion of the artificial exon into the mRNA. Unlike the instant invention, the tagged gene produces a single mRNA (or cDNA) that contains the 5' end of the mRNA, the artificial exon, and the 3' end of the mRNA.

Information Disclosure Statement

The references cited in the JP Search Report issued 12/22/99 have been considered, but will not be listed on any patent resulting from this application because they were not provided on a separate list in compliance with 37 CFR 1.98(a)(1). In order to have the references printed on such resulting patent, a separate listing, preferably on a PTO/SB/08A and 08B form, must be filed within the set period for reply to this Office action. Applicant should omit those references that already have been listed on a PTO-892.

Election/Restrictions

The restriction requirement between groups I and II is hereby withdrawn. Applicant is invited to present new claims directed to group II, since original claims 16-19 have been cancelled. It is suggested that the claims be drafted to avoid the grounds for rejection set forth below. A suggested independent claim directed to the subject matter of original claim 16 is presented below.

Specification

The disclosure is objected to because of the following informalities: Figure 6 discloses SEQ ID NOs: 2-7. However, the specification fails to comply with 37 CFR 1.821(d). Applicant

submitted an amendment to the specification on 6/27/02 directing insertion of an "Appendix". The proposed amendment identified the sequences in Fig. 6 by their assigned SEQ ID NOs, but did not comply with 37 CFR 1.121. As a result, the amendment was not entered. Applicant should resubmit the amendment in compliance with 37 CFR 1.121 to overcome this objection.

Appropriate correction is required.

Claim Objections

Claims 5, 7, 8, 14 and 15 are objected to because of the following informalities.

In claim 5, --a-- should be inserted before "Gal4".

In claims 7 and 14, --a-- should be inserted before "mini-white".

Claims 8 and 15 as amended incorrectly set forth the relationship of the promoter in the gene, and are grammatically incorrect. It is suggested that "is neomycin-phosphotransferase ...promoter" be replaced with -- is a heat shock promoter-directed neomycin phosphotransferase gene --, as described in the specification at page 7, lines 1-5.

Also, in claim 15, -- primary-- should be inserted before "transformants" (line 3), since claim 10 recites primary and secondary transformants. The claim is not indefinite, since claim 15 refers to transformants of step (b), however, reference to "primary transformants, would make claim 15 clearer.

Appropriate correction is required.

Claim 13 is objected to under 37 CFR 1.75(c), as being of improper dependent form for failing to further limit the subject matter of a previous claim. Applicant is required to cancel the

claim(s), or amend the claim(s) to place the claim(s) in proper dependent form, or rewrite the claim(s) in independent form. Claim 10, from which claim 13 depends, requires in step (e) that the secondary transformants be crossed with a fly strain harboring a UAS-luciferase gene. Claim 13 requires performing a different step in place of step (e). Consequently, claim 10 does not embrace the subject matter of claim 13.

Claim Rejections - 35 USC § 112

Claims 1-3, 5-7, and 10-14 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Claims 1 and 10 have been amended (Reply of 9/29/03) to recite "promoter directed drug resistance gene". This is new matter. The original specification and claims describe a "drug resistance gene" and a "heatshock promoter directed neomycin-phosphotransferase gene" (page 7). The original specification does not describe a generic "promoter directed drug resistance gene." The specification is silent on whether the generic "drug resistance gene" is linked to a promoter within the vector, as opposed to being expressed by a trapped promoter, for example. While it may be obvious from the specification that one could operably link a generic drug resistance gene to a promoter in the vector, "[I]t is not sufficient for purposes of the written description requirement of Section 112 that the disclosure, when combined with the knowledge in the art, would lead one to speculate as to modifications that the inventor might have

envisioned, but failed to disclose." Lockwood v. American Airlines Inc., 41 USPQ2d 1961, 1966 (CAFC 1997). Given the lack of description of any subgeneric embodiment between a genus of "drug resistance gene" and the species "heatshock promoter directed neomycin-phosphotransferase gene," it is suggested that claims 1 and 10 be limited to the latter, with cancellation of claim 8, which would be a duplicate of claim 1. Claim 14 need not be cancelled, because the G418 selection is not required by claim 10.

Claims 2 and 11 have been amended (Reply of 9/29/03) to recite that the plasmid is made by inserting the drug resistance gene into pCasper3, rather than simply being derived from pCasper3 as originally claimed. This is new matter. The vector pCasper is a pUC based plasmid comprising a recombinant P-element, which contains a mini-white gene and a polycloning site upstream of the mini-white gene. The mini-white gene comprises the white gene promoter and transcription termination sequences. Inserting a drug resistance gene into it will not produce the vector recited in claim 1. Also, the original specification (pages 6-7) only describes making a single type of vector starting with pCasper3. The specification describes inserting a sequence consisting of, in order: an artificial splice acceptor site; a "stop/start" sequence; and a promoterless GAL4 gene into the polycloning site of pCasper3. Next, the entire 3'UTR of the mini-white gene is replaced with an artificial splice donor site. The resulting plasmid vector, presumably pTrap, is not pCasper3. Finally, a heat shock promoter-directed neomycin phosphotransferase gene is inserted into the plasmid. pTrap-hsneo, shown in Fig. 1 and SEQ ID NO: 1, is an example of such a construction. The original specification does not generically teach a vector that is made by insertion of a drug resistance gene into pCaster3, nor does it teach a vector based upon pCaster3 in which a genus of drug resistance gene is inserted. This part of the

rejection would be overcome by replacing "the promoter directed drug resistance gene into pCasper3" with:

--a sequence consisting of the an artificial consensus splicing acceptor site, the synthetic stop/start sequence, and a promoterless Gal4 reporter gene into the polycloning site of pCasper 3; then replacing the entire 3'UTR of the mini-white gene of pCasper3 with a synthetic splicing donor site; and finally, inserting a heat shock promoter-directed neomycin phosphotransferase gene between the promoterless Gal4 reporter gene and the mini-white gene--.

Claims 1-3, 5-8, and 10-15 are rejected under 35 U.S.C. 112, first paragraph, as based on a disclosure which is not enabling. The inclusion of the "nucleotide sequences" in a P-element, which is critical or essential to the practice of the invention, but not included in the claim(s) is not enabled by the disclosure. See *In re Mayhew*, 527 F.2d 1229, 188 USPQ 356 (CCPA 1976).

The specification (page 4, lines 2-4; page 8, line 20, to page 9, line 9; page 10, lines 11-18; page 10, line 27 to page 11, line 16) and claim 10 indicates that the vector is introduced into a fly to produce a first transformant, which is then crossed to a fly harboring a transposase. The transposase then causes the "vector" to jump to new locations. The invention is directed to new locations that are in genes. The specific examples described in the specification are all vectors that comprise the "nucleotide sequences" in a P-element. The only way a transposase can cause the "vector" to jump to a new location is if the "vector" where a transposon. The specification does not mention transposons generally, but rather describes only P-elements. It is also not accurate to say that the "vector" jumps, as in claim 10. What "jumps" is the P-element that was contained on the vector. Consequently, although the specification does not explicitly state that

the "nucleotide sequences" must be contained in a P-element on the vector, the disclosure when taken as a whole implicitly teaches that the core of the invention is a new type of recombinant P-element, i.e. the P-element is implicitly disclosed as essential.

Claims 1-3, 5-7, and 10-14 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for embodiments where the drug resistance gene encodes neomycin phosphotransferase, does not reasonably provide enablement for other drug resistance genes. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The claims broadly require a "drug resistance gene". As disclosed in the specification (e.g. page 4, line 1, and page 7, lines 1-6), the purpose of the "drug resistance gene" is to confer resistance to killing of transformed flies by the drug. The drug will then kill flies that do not carry the drug resistance gene, and allow the practitioner to select flies transformed with the vector from a population that contains non-transformed flies. In order to practice the invention, two products are required: first, a drug that kills *Drosophila*; and second, a gene that when expressed in *Drosophila* will prevent killing by the drug. The only combination of drug and gene disclosed in the instant specification that meets these requirements are G418 and a gene encoding neomycin phosphotransferase. There is no evidence of record that any other combination of drug and drug resistance gene meeting these requirements was known in the prior art. Consequently, one of skill in the art would be required to identify drugs that kill *Drosophila* and also a gene that when expressed in *Drosophila* would confer resistance to that drug. The specification provides

no guidance as to other likely drugs or drug resistance genes that would be suitable, or how one would go about identifying any such combinations. The specification must teach those of skill in the art how to make and how to use the invention as broadly claimed. *In re Goodman*, 29 USPQ2d at 2013 (Fed. Cir. 1994), citing *In re Vaeck*, 20 USPQ2d at 1445 (Fed. Cir. 1991).

Claims 1-3 and 5-15 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 1-3, 5-8, and 10-15 are indefinite for recitation of "reporter gene" and "gene responsible for a detectable phenotype". It is unclear what functional elements are present or absent in a "nucleotide sequence" recited in the claims as being a "gene". The term "gene" is not defined in the specification, and it does not have a single agreed upon meaning in the art. In the art, gene can mean anything from just the coding sequence for a protein, genomic sequences that are transcribed to produce mRNA, and up to all sequences required for transcription and translation of a gene product, including the promoter. Consequently, one must infer the meaning of "gene" in the claims from the way it is used in the specification. However, the specification is inconsistent in what "gene" is used to describe. The claims recite at least three different genes: a reporter gene, a drug resistance gene, and a gene responsible for a detectable phenotype. With respect to reporter gene, it is clear from the specification that "gene" here does not include the promoter, e.g. page 6, lines 21-26, or transcription termination signals, e.g. Fig. 1, and Fig. 5, i.e. "gene" here means only coding sequence. With respect to "drug resistance gene," it is unclear if this "gene" includes a promoter or not. Original claim 8 recited "drug resistance gene ... its

promoter is" as if the promoter was included as part of the drug resistance gene, but page 7 in reciting "heatshock promoter directed neomycin-phosphotransferase gene" appears to refer to "promoter" and "drug resistance gene" as being separate elements. The term "promoter directed drug resistance gene" is definite, but as indicated above this subgenus of "drug resistance gene" is not described in the original specification and is new matter. It is clear that "gene" here definitely includes the coding sequence, but it is unclear whether a promoter is part of "gene" or a separate element. Finally, with respect to the "gene responsible for a detectable phenotype," there is no discussion whether the genus of this gene includes a promoter or not, or a transcription terminator or not, although it is clear from the specification that no transcription termination sequences should be linked to this gene in the vector.. However, in describing the mini-white gene, it is clear that the "mini-white gene" includes not only the coding sequence, but also its own promoter and transcription termination signals. Fig. 5 describes the mini-white gene as "being expressed itself," which implies that the gene comprises a promoter, and Fig. 1 shows the m-white promoter. Page 6, lines 27-30, describes removing the transcription termination signals from the mini-white gene, i.e. "gene" as applied to the mini-white gene includes not just the coding sequence, but the promoter and transcription termination sequences as well. However, it is not clear if the genus "gene responsible for a detectable phenotype" includes the promoter.

Claim 3 recites the limitation "the Gal4 gene" in lines 1-2. Claim 6 recites the limitation "the Gal4-firefly luciferase fusion gene" in lines 1-2. Claim 9 recites the limitation "the pCasterhs" in lines 2-3. Claim 10 recites the limitation "the cDNAs" in lines 1-2. Claim 12 recites the limitation "the Gal4 gene" in line 2. Claim 13 recites the limitation "the Gal4-firefly luciferase fusion gene" in line 2. Claim 14 recites the limitation "the cDNAs" in line 3. Claim 15

recites the limitation "the transformants resistant to G418" in line 3. There is insufficient antecedent basis for these limitation in the claims. In claims 3, 6, 9, 12, 13, "the" should be replaced with -- a --; in claims 10 and 14, "the" should be deleted.

Claim 10 recites the limitation "the flies" in step (d). There is insufficient antecedent basis for this limitation in the claim. The claim recites at least four different flies: the white minus fly (a), the primary transformants (b), the transposase source strain (c), and the secondary transformants (d), none of which are explicitly referred to as "flies." See suggested claim 10 below for remedial language.

Claim 10 recites the limitation "the reporter gene expression" in step (e). There is insufficient antecedent basis for this limitation in the claim.

Claims 10-15 are rejected under 35 U.S.C. 112, second paragraph, as being incomplete for omitting essential elements, such omission amounting to a gap between the elements. See MPEP § 2172.01. The omitted elements are: that the "nucleotide sequences" are contained in a recombinant P-element in the plasmid, that the reporter gene is a promoterless Gal4 gene, and that the gene responsible for a detectable phenotype in *D. melanogaster* is a mini-white gene.

With respect to the P-element, step (c) requires that the "vector" be forced "to jump to other locations" by the transposase. The "vector" must be a transposon for this to occur. The only transposon described in the specification is a P-element, and indeed all the specific vectors described carry P-elements.

With respect to the reporter gene, step (e) requires measuring expression of the reporter gene when present in a fly with a UAS-luciferase strain. The UAS requires Gal4 for expression,

and implicitly, the reporter expression is measured indirectly by measuring luciferase expression. This grounds of rejection does not apply to claim 13. Also, the reporter gene is used to trap a promoter. If it has a promoter, then it cannot be used this for this purpose. The specification makes clear that the reporter gene should be promoterless. This applies to claim 13.

With respect to the gene responsible for a detectable phenotype, step (a) requires that the primary transformant be made in a white minus fly and step (d) requires that secondary transformants be selected on the basis of strong eye color. The only way the limitation of step (d) can be met is if the gene responsible for a detectable phenotype is one that confers strong eye color in a white minus background, e.g. a mini-white gene. Since the only such gene disclosed is a mini-white gene, this gene in the claims should be limited to the mini-white gene. Otherwise, new matter would be introduced in that the specification does not describe a genus of gene that confers eye color in a white minus background.

Allowable Subject Matter

Claim 9 would be allowable if rewritten or amended to overcome the rejection(s) under 35 U.S.C. 112, 2nd paragraph, set forth in this Office action.

Claims 1 and 10 would be allowable if amended as follows. Limiting the "gene responsible for a detectable phenotype of the *Drosophila melanogaster*" to the mini-white gene is suggested to overcome the rejections under §112, 2nd para. without introducing new matter, similar to the new matter introduced by amending claim 1 to recite "promoter directed drug resistance gene." The original specification does not describe a generic "gene responsible for a detectable phenotype" under control of its own promoter or a heterologous promoter.

Furthermore, limitations in the method imply that a white gene is required to practice the method, e.g. step (d). Limiting the reporter gene in claim 10 to Gal4 is suggested because claim 13 does not further limit claim 10, and because step (e) is inoperative if the reporter gene does not encode Gal4. Expression of the luciferase requires Gal4.

Claim 1 (currently amended). A vector for trapping an unknown gene of *Drosophila melanogaster*, which is a recombinant plasmid comprising a recombinant P-element, wherein the P-element comprises the following nucleotide sequences in this order:

an artificial consensus splicing acceptor site;

a synthetic stop/start sequence;

a promoterless reporter gene;

a heatshock promoter directed ~~drug resistance~~ neomycin phosphotransferase gene; and

a ~~gene responsible for a detectable phenotype of the *Drosophila melanogaster*~~ a mini-white gene under control of a white gene promoter [[;]] and comprising a synthetic splicing donor site in place of a poly-A addition site.

Claim 10 (currently amended): A method for trapping an unknown gene of *Drosophila melanogaster* by using a vector which is a recombinant plasmid comprising a recombinant P-element, wherein the P-element comprises the following nucleotide sequences in this order:

an artificial consensus splicing acceptor site;

a synthetic stop/start sequence;

a promoterless Gal4 reporter gene;

a heatshock promoter directed ~~drug resistance~~ neomycin phosphotransferase gene; and

a ~~gene responsible for a detectable phenotype of the *Drosophila melanogaster*~~ a mini-white gene under control of a white gene promoter [[;]] and comprising a synthetic splicing donor site in place of a poly-A addition site,

which method comprises the steps of:

(a) introducing the vector into the genome of a white minus fly;

(b) selecting primary transformants containing the vector;

(c) crossing the primary transformants with a transposase source fly strain to force the P-element ~~vector~~ to jump into other locations;

(d) selecting secondary transformants by ~~picking up the~~ selecting flies produced from the cross of step (c) having strong eye color;

(e) crossing the secondary transformants with UAS (Upstream Activator Sequence)-luciferase harboring fly strain and measuring ~~the reporter gene~~ expression of the reporter gene in the secondary transformants ~~the resultant flies~~; and

(f) identifying the trapped gene by cloning and sequencing cDNA comprising the Gal4 gene and cDNA comprising the mini-white gene ~~the cDNAs fused to the reporter gene and the gene responsible for a detectable phenotype of the fly~~.

The following independent claim is suggested, should Applicant wish to add claims directed to group II, which has been rejoined. Limiting the claim to the mini-white gene is suggested for the same reasons as for suggested claim 10.

20. (New) A method for trapping an unknown gene of *Drosophila melanogaster* by using a vector A and a vector B;

wherein vector A is a recombinant plasmid comprising a recombinant P-element, wherein the P-element comprises the following nucleotide sequences in this order:

an artificial consensus splicing acceptor site;

a synthetic stop/start sequence;

a promoterless Gal4 DNA binding domain-P53 fusion gene as a reporter gene;

a heatshock promoter directed neomycin phosphotransferase gene; and

a mini-white gene under control of a white gene promoter and comprising a synthetic splicing donor site in place of a poly-A addition site and

vector B is derived from pCasperhs by inserting a heatshock promoter directed Gal4 activator domain-large T antigen fusion gene within polycloning site of the pCasperhs,

which method comprises the steps of:

(a) introducing each of the vectors A and B into the genomes of separate white minus flies;

(b) selecting primary transformants for the vector A which are resistant to G418 and selecting primary transformants for the vector B which have an eye color other than white;

(c) crossing the primary transformants for the vector A with a transposase source fly strain to force the P-element to jump into other locations;

(d) selecting secondary transformants for the vector A by selecting flies produced by the cross of step (c) that have strong eye color;

(e) crossing the secondary transformants with the primary transformants for the vector B to obtain flies harboring the P-element and vector B;

(f) crossing the flies obtained in the step (e) with an UAS-luciferase harboring fly strain and measuring luciferase expression of the resultant flies after a heatshock treatment; and

(g) identifying the trapped gene by cloning and sequencing cDNA comprising the reporter gene and cDNA comprising the mini-white gene.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Scott D. Priebe, Ph.D. whose telephone number is (571) 272-0733. The examiner can normally be reached on M-F, 8:00-4:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on (571) 272-0735. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Scott D. Priebe

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Art Unit 1632